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STUDIES ON PIGLET MORTALITY

1. Streptococcal Meningitis and Arthritis

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INTRODUCTION

Streptococcal infection of the central nervous system has been reported in swine both in Europe and North America. Gasperi & Grippi (1912) and Lesbouyries (1932) in France, Ehrlich (1926) and Glässer (1944) in Germany, Ray (1945) in the U.S.A., and Jensen & van Dorssen (1951) in Holland, have described cases of meningitis in pigs in which streptococci were incriminated. The latter authors were able to reproduce the condition experimentally by the intravenous or intra-cerebral inoculation of cultures of the streptococci isolated from naturally occurring cases.

Although this disease has not been reported in pigs in Great Britain, experience over the last few years has persuaded us that there is in this country a fairly well defined syndrome in piglets associated with a streptococcal infection. The most common form of the disease is characterised by ataxia and partial or complete inability to maintain balance due to meningo-encephalitis resulting from invasion of the brain and its meninges. Infected piglets do not, however, necessarily develop nervous symptoms. Some recover spontaneously, others die during the bacteraemic stage of infection and in some cases infection is followed by invasion of the joints, particularly those of the stifle, hock and knee, giving rise to arthritis. All forms of the disease can usually be recognised in an infected herd and on occasion in a single litter. The present paper is an attempt to summarise the observations made on a number of outbreaks investigated during the last few years.

CLINICAL ASPECTS

The first cases to be diagnosed occurred in a litter of cross-bred piglets on an Essex pig farm, since when the disease has been recognised in other herds in Essex, Suffolk, Norfolk, Cambridge, Berkshire and Oxfordshire. Some idea of its incidence can be gathered from the fact that between January and June, 1954, outbreaks were diagnosed on 11 farms in East Anglia. The disease appears to be confined to piglets between two and six weeks of age. Its incidence varies considerably in different herds and in individual litters. In some herds clinical symptoms of infection have appeared in five or six successive litters, whereas in other herds the disease may be confined to a single litter. Sometimes an individual piglet in a litter is affected ; in other cases most of the piglets show clinical symptoms. Outbreaks have occurred at all times of the year and in herds kept under various methods of management. One outbreak was diagnosed on a farm where the sows and litters were kept at pasture throughout the year in fold units, each unit being separated by electric fencing. Other outbreaks have occurred in litters born and reared in permanent farrowing units. Housing appears to play little or no part in the spread of infection, for once the disease is established in a herd, symptoms may appear in litters born and reared in buildings which have not been used previously for the housing of pigs.

In the majority of outbreaks the early signs of infection are elevation of temperature and anorexia, the piglets burying themselves in the bedding for warmth. If disturbed and moved round the pen, one or more show a swaying movement of the hind quarters and/or a peculiar stiff-legged gait, giving the appearance of walking on the toes. Affected piglets in one litter were observed to stretch themselves by extending the forelegs forward and crouching like dogs. The ears are often retracted and held close to the side of the head. There is frequently some interference with sight and some piglets show a violent shaking of the whole body, particularly after forced exercise. Subsequent symptoms are extremely variable. Some infected piglets die during the febrile stage of illness. In other cases, swaying of the hind quarters is followed by complete inability to maintain balance, affected piglets lying on one side and " paddling " with all four legs. Death sometimes follows from hypostatic pneumonia. In less severe cases ataxia does not proceed beyond the stage of swaying of the hind-quarters while a few mild cases recover spontaneously. In some protracted cases swellings appear over the stifle and around the hock joints, due to the distension of the joint capsules with fluid. Piglets recovering from infection fail to thrive and joint swellings, when present, often persist for several months. In early cases of infection a response has followed treatment with penicillin. More advanced cases showing nervous symptoms generally fail to respond, or, if responding, may relapse in 7 to 10 days, probably due to the compression of the brain and nerve roots by fibrous tissue.

The results of cultural examination depend upon the stage of the disease at which the examination is made. If piglets die during the febrile stage of infection, the streptococcus is invariably isolated from the visceral organs. Invasion of the brain or joints nevertheless occurs quite early, as one or other of these may also yield positive cultures. In the later stages positive cultures may be obtained only from the brain and/or joints. The disease is, therefore, primarily a bacteraemia with subsequent invasion of the central nervous system and/or the joints. Table I gives the cultural results from representative cases from seven outbreaks of the disease. Six of the seven piglets in this series were slaughtered when showing clinical evidence of infection.

Table I
Cultural Examination of Seven Piglets with Streptococcal Infection

	Case 1	Case 2	Case 3	Case 4	Case 5	Case 6	Case 7
Brain	+	+	+	+	C	N.C.	Neg.
Visceral Organs	+	Neg.	Neg.	Neg.	+	+	+
Joints	N.C.	+	Neg.	+	+	Neg.	+

+ = Positive in culture for streptococci.
C = Cultures contaminated.
N.C. = Not cultured.

Characteristics of the Organism

A streptococcus having the following characteristics has been isolated from all outbreaks of the disease.

Morphology

In smears prepared from the cerebro-spinal fluid or ventricular pus the organisms appear as gram-positive cocci, occurring singly or in pairs ; chains are rare and always short.

Sheep Blood Agar

When grown on sheep blood agar the colonies are surrounded by a zone of α haemolysis.

Horse Blood Agar

Colonies are surrounded by a clear zone of β haemolysis.

Edwards's Crystal Violet Aesculin Blood Agar

The streptococci split the aesculin and appear as black colonies surrounded by a zone of greening.

Broth

A poor growth is obtained in nutrient broth. Growth is improved by the addition of 10 per cent. horse serum, the organism producing a uniform turbidity.

Biochemical Reactions

Acid, no gas, is produced in trehalose, salicin, inulin, lactose, dextrose, maltose, laevulose, sucrose and galactose : no change in sorbite, mannite, raffinose, arabinose, dulcite, glycerin, inosite, rhamnose and xylose. Some strains produce partial reduction in 1 : 5,000 methylene blue milk. Acid is produced in Litmus milk. Starch is hydrolysed.

Serological

The organism does not yield an extract that gives a precipitate with any of the Lancefield group sera, including that for group P. A serum prepared in rabbits against one strain gave a weak precipitin reaction with extracts prepared from eight other strains isolated from different outbreaks, indicating that the strains are antigenically similar. This serum did not give a precipitate when tested against extracts of all the Lancefield groups, including a number of Group P streptococci isolated from pigs. Six strains of the streptococcus have been examined by Dr. R. E. O. Williams at the Streptococcal Reference Laboratory, Colindale, who confirms that the organism has an antigen that is different from those characteristic of any of the previously recognised Lancefield's groups.

Pathogenicity

Biological tests using broth cultures of two strains isolated from separate outbreaks show that the organism is non-pathogenic for mice, guinea-pigs and rabbits by subcutaneous, intraperitoneal, intravenous and intracranial inoculation. One rabbit injected subcutaneously with 1 ml. of a 1 : 5 suspension of infected brain tissue in sterile distilled water remained healthy.

Pathogenicity for Pigs—Experimental Reproduction of the Disease

The disease has been reproduced in piglets both by the intravenous and subdural inoculation of broth cultures of the streptococcus described in the preceding section.

Intravenous Route of Infection

Four 24-day-old litter-mates were given an intravenous injection of a 24-hour broth culture, the individual doses being 1, 2, 3 and 4 ml. respectively. The piglets were housed in separate wire cages for the period of the experiment. Two of the piglets showed febrile reactions with rectal temperatures of 107° F. on the fifth day following inoculation. One of these piglets developed a typical nervous syndrome three days later, being unable to maintain balance and lying on its side paddling all four limbs vigorously. When killed on the 11th day following injection the streptococcus was isolated in culture from the brain and joints but not from the visceral organs. The second piglet showed no nervous symptoms but gradually lost condition. When killed 13 days after experimental infection a few streptococci were isolated from the brain and cerebro-spinal fluid and a heavy growth from the joints. The remaining piglets remained clinically normal but when examined 19 days after injection the hock joint of one yielded a growth of the streptococcus.

Subdural Route of Infection

Two store pigs were each inoculated subdurally (under nembutal anaesthesia) with 1 ml. of a serum broth culture of a streptococcus isolated from a field case. One of the pigs developed a progressive meningo-encephalitis and was killed *in extremis* on the sixth day following infection. Early symptoms were dullness, stiffness of the neck, intermittent nystagmus and occasional muscular twitching. Later on the symptoms regarded as being typical of a field case developed, the piglet lying on one side and making continuous paddling movements with all four legs. Nystagmus was evident and the tone of the squeal altered. The pig was destroyed and at *post-mortem* examination showed lesions in the brain similar to those found in field cases. The second pig, although dull and off its food, showed no nervous symptoms other than a slight ataxia and stiffness of the neck. *Post-mortem* examination on the sixth day showed a slight fibrino-purulent meningitis. No lesions were found in the nervous tissue. The results obtained on cultural examination of the experimentally infected piglets are shown in the following table.

Table II
Results of Cultural Examination of Experimentally Infected Piglets

	Intravenous route				Subdural route	
	Pig A 11 days	Pig B 13 days	Pig C 19 days	Pig D 19 days	Pig A 6 days	Pig B 6 days
Brain	+	+	Neg.	Neg.	+	+
Visceral organs	Neg.	Neg.	Neg.	Neg.	Neg.	+
Joints	+	+	+	Neg.	N.C.	N.C.

+ = Positive in culture for streptococci.
N.C. = Not cultured.

H. I. FIELD, D. BUNTAIN & J. T. DONE: STUDIES ON PIGLET MORTALITY

I.—Streptococcal Meningitis and Arthritis

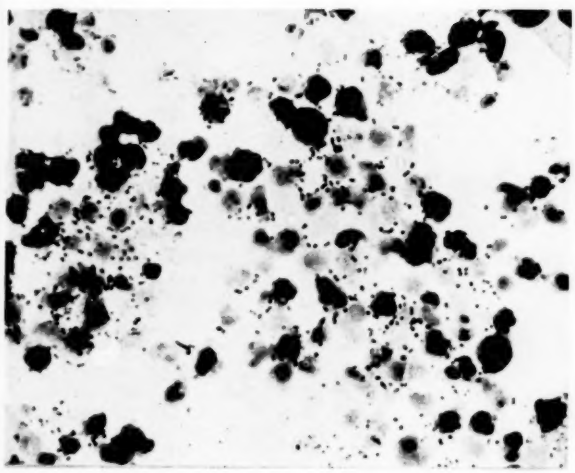

FIG. 1.—Field case. Smear of pus from the fourth ventricle. Stained Gram. × 780. The purulent nature of the exudate and the characteristic forms of the organism in the tissues can be seen.

FIG. 3.—Field case. Section through the fourth ventricle. Stained H. and E. × 240. The *tela choroidea* is distended by cellular exudate and there are accumulations of pus lying free in the ventricle.

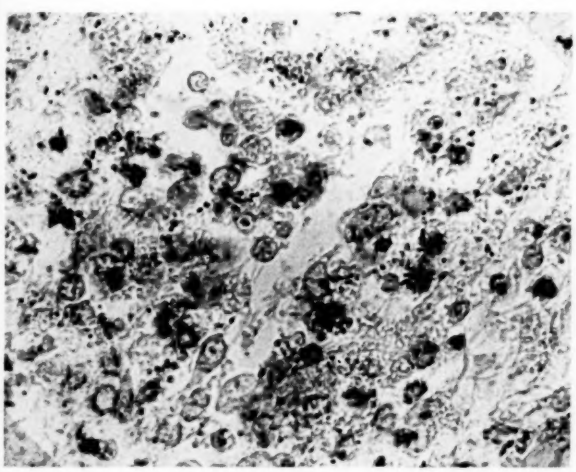

FIG. 2.—Field case. Section through choroid plexus of the fourth ventricle showing bacteria intra- and extra-cellularly. Stained Gram. × 780.

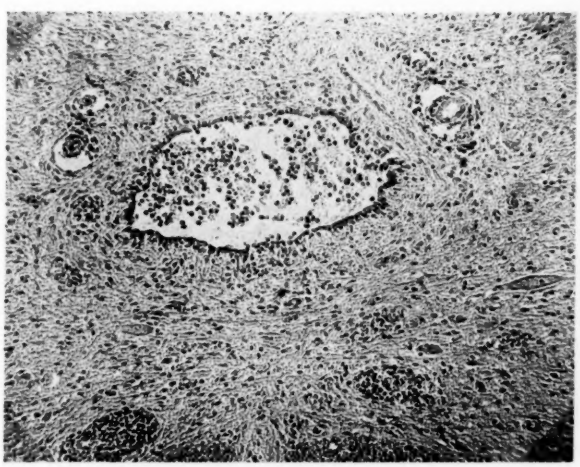

FIG. 4.—Experimental case, killed 11 days after intravenous inoculation of culture. T.S. Lumbar Cord. Stained H. and E. × 240. The central canal contains fibrino-purulent exudate and many of the vessels in the grey commissure are cuffed.

Pathology

In cases showing paraplegia or complete inability to maintain balance the only macroscopic abnormalities consistently present at *post-mortem* examination were to be found in the brain and cord. The cerebro-spinal fluid, usually under increased pressure, was slightly turbid to opalescent and in some cases distinctly creamy. The blood vessels of the brain and meninges were congested and in the subarachnoid space there was often an accumulation of whitish pus giving an abnormal opacity to the leptomeninges. The choroid plexuses of the brain were generally severely affected, especially that of the fourth ventricle which appeared opaque, light yellow in colour and of the consistency of cream. In some cases the ventricles, aqueduct and the central canal of the medulla and cord were completely blocked by exudate, and a non-communicating type of hydrocephalus was present as a result of the obstruction to the flow of C.S.F.

Smears from the ventricular pus showed a highly cellular exudate in which macrophages (many packed with ingested cells as well as bacteria) predominated. Bacteria were seen intracellularly in large numbers and also free.

Histologically the picture was usually one of an acute fibrino-purulent choriomeningitis with secondary extension to the subpial and periventricular tissues. The meningeal exudate usually invested the roots of the cranial and at least the more anterior spinal nerves. The fourth ventricle was the most often and most severely involved of the ventricles possibly because by virtue of its position it is liable to infection from either the meninges or the ventricular system, but, though the anterior ventricles were always involved to some extent, extension to the spinal cord by way of the central canal was more variable. Though essentially an infection of the meninges and tela choroidea some cases showed extensive liquefactive necrosis of the nervous tissue proper, especially the cerebellum, brain stem and cervical cord. In fulminating infections cocci were present in great numbers in the necrotic tissues, but in some less acute cases they were difficult to demonstrate in sections though recoverable in pure culture by bacteriological methods. So far we have not had an opportunity to examine the brain from a pig which has survived after showing clinical disease, but it is possible that at least some of the few cases of chronic arachnoiditis in store pigs seen by one of us (J. T. D.) may represent such animals.

The pathological changes found in the brains of experimentally infected piglets were similar to those occurring in natural cases. Of the two pigs experimentally infected by subdural inoculation, one showed lesions virtually indistinguishable from a field case, while the other showed less pronounced lesions of fibrino-purulent meningitis. In the case of the piglet which developed a typical nervous syndrome following intravenous injection of culture, the pathological changes were again identical with those found in natural cases of the disease.

In the arthritic form of the disease, the joint fluid was invariably turbid and showed a high cell content, although only a few streptococci might be present. Piglets dying during the febrile stage of the illness generally showed an excess of peritoneal fluid from which the organisms were sometimes isolated in culture, but there were no diagnostic lesions in the visceral organs.

Discussion

Streptococcal infections are comparatively common in pigs of all ages. The type isolated most frequently is a beta haemolytic organism which serologically falls into Lancefield's Group C. This streptococcus is associated with a variety of pathological conditions but its most common habitat appears to be the porcine uterus, where it is frequently found, sometimes associated with metritis. The streptococci isolated from our outbreaks of meningitis do not fall into any of Lancefield's groups and we have not found them associated with any other pathological condition in pigs. Cotchin & Hayward (1953) reported the isolation of a somewhat similar streptococcus from a case of endocarditis in a pig. The intravenous injection of a culture of their organism into experimental pigs gave rise to endocarditis and arthritis but no nervous symptoms were shown by any of the infected pigs. No evidence of endocarditis was found in any of our cases so that it seems doubtful whether they were dealing with the same organism as described in the present report. The observations that housing appears to play little or no part in the spread of infection and that the disease is confined to piglets between two and six weeks of age point to the sow as the probable source of infection.

The disease presents little difficulty in diagnosis. In most infected litters several piglets show clinical symptoms characterised by lack of balance or early symptoms of arthritis. This serves to differentiate it from the sporadic cases of purulent meningo-encephalitis or arthritis which are sometimes encountered. The former usually result from an extension of infection from the nasal cavity or middle ear, the bacteria isolated in culture from such cases being usually *C. pyogenes*, *Pasteurella* or *Pseudomonas*. The sporadic cases of arthritis are generally due to infection with a Lancefield's Group C streptococcus, although occasional cases may be met in which the joints are infected with *Haemophilus suis*. Another differential characteristic is that there is no reddening of the skin as may occur in acute swine fever or erysipelas infection.

The main diagnostic features at *post-mortem* examination of cases showing nervous symptoms are the excess of highly cellular cerebro-spinal fluid and the accumulation of purulent material in the subarachnoid space. Smears show macrophages, polymorphs and organisms.

Summary

Streptococcal meningitis and arthritis is a specific clinical entity occurring in piglets between two and six weeks of age. It is characterised in some cases by partial or complete inability to maintain balance and in others by suppurative arthritis. Early cases of infection respond to treatment with penicillin, to which the streptococcus is sensitive by *in vitro* tests. The streptococcus, which can be isolated from all outbreaks of the disease, does not fall into any of the Lancefield's groups. It is alpha haemolytic on sheep blood agar, beta haemolytic on horse blood agar and splits aesculin. The disease can be reproduced in piglets by the intravenous or subdural injection of culture. Infection may be confined to a single litter in a piggery, although in some herds the disease may appear in successive litters over a period of many months. Housing appears to play little or no part in the spread in infection.

Acknowledgments.—We are indebted to Mr. G. Weston, F.I.L.M.T., for photographs of Figs. 1 and 2.

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DUCK VIRUS HEPATITIS

BY

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Levine & Fabricant (1950) reported that in 1949 a disease appeared on duck farms on Long Island which was different from anything that experienced duck farmers had seen before. At first, two- and three-week-old ducklings were affected, but as time passed younger ducks became affected until even three-day-old ducklings were involved. The disease spread so rapidly that within six months practically every one of the 75 duck growers in the area had reported losses.

The mortality varied considerably from farm to farm and even from hatch to hatch on the same farm.

In severe outbreaks the losses were as high as 85 to 95 per cent. in some hatches. At the beginning, every brood of ducks contracted the disease. As time passed an occasional batch would escape with practically no mortality. Sometimes several batches seemed to be free from the disease, but it always reappeared in later batches.

The disease was shown to be due to a filterable virus and the term Duck Virus Hepatitis was applied to it.

Duck Virus Hepatitis in Norfolk

In July, 1953, a disease appeared in one- to two-week-old ducklings on a farm in Norfolk. The losses commenced in ducklings purchased as day-olds from another duck breeder in Norfolk and quickly spread to home-hatched ducklings housed in adjoining brooders.

The disease ran a rapid, violent course, with most of the losses on the second or third day of the outbreak. The survivors rapidly recovered. The mortality varied from batch to batch and was sometimes as high as 60 per cent.

After emptying the brooder house of ducklings and cleaning the brooders, the first batch of day-old ducklings introduced sickened during the second week. From these ducklings the first isolations of virus were made. It should be pointed out that survivors from earlier affected groups and chickens were still on the farm in pens not far distant from the brooder house. No unusual losses were reported in 20,000 growing or adult ducks present at this time. During September, hatching was suspended and the brooder house and equipment were cleaned and disinfected. Two weeks later the owner commenced rearing day-old chicks in the brooder house and followed these at the beginning of November with day-old ducklings. There was no loss in the first batch of 70 ducklings from home-produced eggs. During each of the next three weeks, however, losses occurred in batches of ducklings of mixed origin, *i.e.*, containing both home-produced and purchased ducklings. The mortality varied from 3 to 7 per cent. Some of the survivors appeared sleepy, huddled up and disinclined to move about. Virus was again isolated from some of these ducklings. During the Christmas period the losses were reported to be heavy ; but were less serious the following week, only to increase the next week when virus was again isolated.

All brooder houses were again emptied, cleansed and disinfected. All day-old ducklings were moved direct from the hatchery to a neighbouring farm. Here they were reared until three to four weeks old, when they were brought back to the premises and moved into outdoor runs. No unusual losses were experienced. About the middle of March, some of the three-week-old ducklings were moved into the brooder house. After four to five days deaths again occurred and virus was isolated.

This outbreak of disease is the first known case of its kind in Britain.

The onset of symptoms both in naturally and artificially infected ducklings was very rapid. Some ducklings were found dead without symptoms having been observed. More often the first signs of disease were general weakness and dullness, affected ducklings being unable to keep up with the others. If knocked over they had great difficulty in regaining their feet. Affected ducklings rapidly lost interest in food. As the condition advanced they would be found lying on their sides or breasts with legs extended backwards and head drawn over the back.

The incubation period in ducklings artificially infected when one to three days old varied from two to seven days ; most of the ducklings sickened on the second or third day.

Recovery was sometimes remarkably rapid, individual ducklings which appeared very sick, might, a few hours later, look apparently unaffected. Once the crisis was passed survivors usually formed an even group not noticeably lighter than uninfected controls.

In some affected ducklings no gross lesions could be seen. In others the most conspicuous lesions were found in the liver. It was usually enlarged and contained punctuate or ecchymotic haemorrhages. These were usually few and widely scattered but in some individuals they were very numerous and often confluent. In some birds the liver surface showed red, blotchy discolorations, without definite haemorrhages.

The spleen was enlarged and pale in some ducklings and the kidneys were swollen. In some artificially infected ducklings the myocardium was cream coloured. Cultures made on sheep blood agar with heart, blood, liver, spleen and bile, from recently dead ducklings yielded no bacterial growth.

Transmission to Chick Embryos

Lesions resembling those described by Fabricant & Levine (1950) were produced with regularity in chick embryos inoculated with filtrates, with bacteriologically sterile crude liver and spleen suspensions and with suspensions treated with penicillin and streptomycin.

The character of the lesions and their course depended on the age of the embryo at inoculation. The most marked and characteristic lesions were seen in embryos inoculated into the chorio-allantoic sac after 10 or 11 days' incubation. Only a few embryos died in the three days following inoculation, some of these were oedematous, others showed no gross abnormality.

On candling the eggs from the end of the third day after inoculation onwards, the embryos appeared sluggish or were completely inert. The chorio-allantoic blood vessels remained readily apparent. Some embryos remained in this state for 8 to 10 days before finally dying.

Embryos dying on the fourth day showed a variety of lesions, usually only a certain number of which were found in any one embryo. Oedema, particularly of the body wall, grossly distended the embryo, giving it a ball-like appearance. The oedema fluid was frequently clear but sometimes was turbid or white. The liver was somewhat swollen with rounded borders. Yellow or white areas of necrosis, sometimes involving the whole of the liver, were frequently present. The necrosis seemed to extend from the surface inwards and was clearly demarcated. The amniotic sac contained an excess of fluid. Except for slow rhythmic heart beats embryo movements were frequently completely lacking.

On the fifth and sixth days similar lesions were present. At this time stunting of the embryo was readily apparent. The liver was frequently stained dark green. Bile pigment was present in the lumen of the intestines and clearly marked

their course. The yolk, particularly at the yolk stalk, and the chorio-allantoic fluid were frequently stained green. This green discoloration could be detected on candling the eggs. Less frequently the heart sac was distended with clear fluid. The spleen was slightly enlarged in some embryos, and in some the heart muscle was cream yellow in colour. The tarso-metatarsal joints and the digits were often fully extended. Most embryos inoculated after seven to nine days' incubation died within four days and apart from generalised oedema might show no gross abnormality.

Embryos inoculated at 12 to 13 days showed similar lesions to those developing in embryos inoculated at 10 days but stunting was less marked, oedema being frequently absent and the green discoloration much lighter in colour. No embryos inoculated after 13 days' incubation or less have hatched, although some were still alive but inert on the 24th day. In the latter the surface of the spleen was sometimes distorted with several whitish seed-like nodules and the caecae were distended with dark green fluid.

The hatchability of embryos inoculated after 15 or 17 days' incubation was reduced and the time of hatching was delayed.

Strong chicks were hatched from all of a group of 12 embryos inoculated after 19 days' incubation. Embryonic fluids of infected chick embryos did not agglutinate the R.B.C.s of ducks or chickens.

Serum Neutralisation Tests

Chorio-allantoic fluid of infected embryos harvested on the sixth day after inoculation was found to have an Ld_{50} titre of 10,000 to 100,000 per ml.

Serum neutralisation tests were carried out in 10-day embryonated eggs. Equal volumes of decimal dilutions of infective chorio-allantoic fluid were mixed with undiluted serum. Serum-virus mixtures were allowed to stand at room emperature for 30 minutes and then inoculated into the chorio-allantoic sac. Each embryo received 0·1 ml. As a routine 500 units each of penicillin and streptomycin were included in the inoculum for each egg. The reading of titrations was based on the development of gross lesions. Embryos dying during the 48 hours following inoculation were ignored. All embryos alive six days later were opened and examined.

The results of several tests are shown in Table I. The

Table I

Titrations and Serum Neutralisation Test with Duck Virus

Test	Test Mixture Virus+	Virus Dilutions 10^0	10^{-1}	10^{-2}	10^{-3}	10^{-4}	10^{-5}
1	Pooled serum of 10 recovered ducklings	0/3	0/3	0/4	0/4		
	Pooled serum of 10 adult ducks from affected farm	1/4	1/4	0/4	0/4		
	Normal duck serum ...	2/2	2/2	4/4	4/4	1/4	0/4
	Virus titration			4/4	4/4	2/4	0/4
2	Duck virus hepatitis immune serum*	0/5	0/5	0/5	0/5		
	Normal duck serum ...		5/5	5/5	5/5	1/5	0/5
	Virus titration			5/5	5/5	5/5	1/5
3	Louping ill immune sheep serum 1†	3/3	4/4	3/3	0/4		
	Louping ill immune sheep serum 2†	4/4	4/4	4/4	1/4		
	Normal sheep serum ...		3/3	2/3	1/3	0/4	
	Normal duck serum ...		3/3	3/4	4/4	1/4	0/4
	Virus titration		4/4	4/4	3/3	3/4	0/4

Denominator = number of embryos inoculated.
Numerator = number of embryos developing macroscopic lesions.

virus was not neutralised by normal duck serum but the pooled serum of survivor ducklings and duck virus hepatitis immune serum* from America neutralised the virus.

The pooled serum of 10 adult ducks from the affected farm also neutralised the virus. This is of interest since no unusual loss had occurred in these birds since the appearance of disease in the young ducklings. Levine & Fabricant (1950) noted that adult ducks, chickens and turkeys on affected farms did not sicken. The presence of neutralising substances in our ducks suggest that adults may contract an inapparent infection.

Levine & Fabricant (1950) pointed out that the lesions observed in chick embryos inoculated with duck hepatitis virus resembled those described by Burnet (1936) in embryos inoculated with louping ill virus.

Two samples of louping ill sheep immune serum did not, however, neutralise our virus to any greater extent than non-immune sheep serum. All three sera showed some capacity to neutralise this virus when compared with normal duck serum.

Unlike louping ill, duck hepatitis virus failed to infect mice after intracerebral or intraperitoneal injection.

Transmission to Ducklings

Transmission experiments gave variable results. This experience is similar to that recorded by Levine & Fabricant (1950).

In a preliminary experiment, five ducklings, five weeks old, were inoculated intraperitoneally with 0·5 ml. of embryonic fluid from the third chick embryo passage. No symptoms were observed in any of the ducklings but neutralisation tests on the two sera tested showed significant neutralisation. The blood and bile of these five ducklings was not infective to chick embryo when tested 14 days after inoculation.

In a second transmission experiment four groups each of eight two-day-old ducklings were exposed, as shown in Table II, to infective embryonic fluid of the sixth passage. One duckling only in each of groups a, b and d, died. Virus was re-isolated from each of these. Several other ducklings appeared sick on the second and third days, but rapidly recovered. Sera from one duckling from each group were collected 25 days after exposure. Significant neutralisation was shown by the sera of the ducklings which had been inoculated by the intramuscular and intraperitoneal routes, but not by the ducklings fed infective fluid or in contact with inoculated ducklings.

A third test was carried out with 28 ducklings one day old. These were all dosed by mouth with the livers of embryos of the 17th chick embryo passage. The ducklings were then placed in a box and were exposed to a mist of embryonic fluid of the same passage. The ducklings all appeared bright and active on the day following exposure. In the next 24 hours, 13 died and most of the remainder appeared ill. Altogether 18 died and the virus was re-isolated from all. No deaths occurred in 19 uninoculated controls.

In the fourth transmission test, 20 two-day-old ducklings and 30 three-day-old chicks were exposed together to a mist of embryonic fluid of the 22nd chick embryo passage. Ten ducklings died during the next seven days and others appeared ill. Virus was re-isolated from all of those which died. One chicken was found dead on the seventh day and three on the eighth day. No symptoms were observed in the chickens at any time and embryos inoculated with liver suspensions from

* This serum was kindly supplied by Dr. E. Dougherty, Duck Disease Research Laboratory, Eastport, Long Island.

† These sera were kindly supplied by Dr. D. R. Wilson, Animal Diseases Research Association, Moredun Institute, Edinburgh.

TABLE II
TRANSMISSION EXPERIMENTS WITH DUCKLINGS

Experiment	Recipients	Inoculum	Route and Dose	Mortality	Comments
1	5 five-week-old ducklings	Embryonic fluid 5th passage	Intra-peritoneal 0·5 ml.	0	Sera of two ducks tested 14 days after inoculation showed significant virus neutralisation
2	8 two-day-old ducklings 8 " " 8 " " 8 " "	Embryonic fluid 6th passage	(a) intra-peritoneal 0·2 ml. (b) contact with above group (a) (c) intramuscular 0·2 ml. (d) *per os* 0·2 ml.	1 1 0 1	Virus re-isolated from all three dead ducklings. Sera of individual ducklings from Groups (a) and (c) showed virus neutralisations.
3	28 day-old ducklings	Embryonic fluid and liver of 17th passage	Liver *per os* Fluid as mist	18	Most ducklings died or sickened. Sera of three survivors tested showed virus neutralisation. Virus re-isolated from all dead ducklings.
4	30 two-day-old ducklings	Embryonic fluid	Exposed to a mist	10 ducklings	Virus re-isolated from all dead ducklings.
	30 three-day-old chicks	22nd passage		4 chicks	Virus not demonstrated in chicks.

the dead chicks did not develop lesions. All 16 control ducklings remained unaffected, one of 16 control chicks died.

TRANSMISSION TO CHICKENS

Levine & Fabricant (1950) stated that no apparent disease occurred in chicks brooded on affected Long Island farms. In the Norfolk outbreak chicks had been brooded alongside infected ducklings. Although no disease had been reported in the chicks, it was considered that they may have been responsible for maintaining the infection in the brooder house during the period when it was depopulated of ducklings. However, neutralisation tests on the sera of 15 chicks brooded near affected ducklings and 15 pullets reared near pens of ducks gave negative results.

Two small experiments showed that chicks were not solidly resistant to duck hepatitis virus. In the first experiment six two-day-old chicks were fed a mixture of embryonic fluid and liver of the second embryo passage. None of the chicks developed symptoms, buf virus was demonstrated in the faeces on the third but not on the sixth day following dosing. Three chicks were killed on the fifth day. Embryos inoculated with pooled liver suspension of these chicks developed typical lesions. The remaining three chicks were killed on the 15th day but pooled liver suspension did not infect embryos, nor did the pooled serum of these chicks neutralise the duck virus.

In the second experiment nine 15-day-old chicks were inoculated intraperitoneally with fluid of the 11th chick embryo passage. No symptoms were observed in any of the chicks. Individual chicks were killed at the intervals indicated in Table III and suspensions of their livers were inoculated into chick embryos. Virus was present nine days after inoculation and the sera of the two chicks killed on the 14th day neutralised more than 10,000 Ld_{50} of virus.

TABLE III
EXAMINATION OF NINE CHICKS INOCULATED INTRAPERITONEALLY WITH DUCK VIRUS AT 15 DAYS OF AGE

Chick No.	1	2	3	4	5	6	7	8	9
Interval between inoculation and Examination (Days)	1	2	3	4	5	7	9	14*	14*
Virus in liver	+	+	+	+	+	+	+	—	—

* Sera neutralised more than 10,000 Ld_{50}.

DISCUSSION

Little is known of the epidemiology and distribution of duck virus hepatitis. In the United States the disease is of particular importance in young ducklings on Long Island. The Report of the New York State Veterinary College for the year 1951–52 shows that at the Duck Disease Research Laboratory, Eastport, Long Island, virus hepatitis was diagnosed in 47 of 73 consignments of ducklings of up to two weeks of age. In a recent private communication Levine stated that outbreaks had occurred on Cape Cod, Massachusetts and in the western part of New York State about 500 miles distant from Long Island. Recently a report had come to hand which indicated that the disease was present in Illinois.

Two features of the diseases are particularly noteworthy. Firstly, duck virus hepatitis has shown ability to persist and pass from batch to batch. This has been true both in the U.S. and in Norfolk. Secondly, on Long Island the disease spread with rapidity to affect all the duck farms in the area within a short time. The conditions on Long Island are probably more conducive to spread than they are in Norfolk.

Diagnosis should not present any particular difficulty. Sudden mortality in young ducklings unassociated with bacterial disease should lead to suspicion. Levine (1952) considered that liver haemorrhages in young ducklings were practically pathognomonic. Diagnosis would be confirmed by the production of typical lesions in inoculated chick embryos and by neutralisation of the virus with antiserum.

SUMMARY

An outbreak of disease which occurred amongst ducklings on a farm in Norfolk is described.

The course, symptoms and lesions of this disease resembled accounts of duck virus hepatitis in the United States.

A virus was isolated from affected ducklings. This virus was neutralised by duck hepatitis antiserum from the United States.

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THE VETERINARY RECORD
August 7th, 1954

FOOT-AND-MOUTH DISEASE

THE Report of the Departmental Committee on Foot-and-mouth Disease, under the Chairmanship of Sir Ernest Gowers, whose Conclusions and Recommendations we published last week, is a document of considerable public and professional importance. It should have the effect of sweeping away a great deal of uninformed comment—much of it in the popular Press—which has had the effect of suggesting in the public mind that the policy of slaughter was outmoded, and was only persisted in by a prejudiced and wilfully obstructive bureaucracy. As members of the profession would have expected, the Committee has expressed its unanimous opinion that, in the present state of knowledge, and having regard to this country's exceptionally fortunate position in being an island where the disease is not endemic, the policy of slaughter remains the right one, and that " any idea that it would be possible to do away with stamping out by making the whole susceptible animal population—or even all cattle—immune by vaccination is in the realm of fantasy."

Nor was this the Committee's view only. A wide cross-section of expert witnesses, which included delegates from the R.C.V.S., the B.V.A., the National Farmers' Union, the Royal Agricultural Society, the National Cattle Breeders' Association and others, expressed the same opinion. It varied in emphasis between one organisation and another, but of its unanimity there was no doubt. Perhaps more notable still was the opinion expressed by the representatives of overseas countries where the disease is endemic. Their intention, in employing vaccination on a national scale, was to try to reduce the incidence of foot-and-mouth disease to dimensions where a slaughter policy could be effectively employed without disrupting their national agriculture.

Members of the profession will share in our satisfaction at the deserved tribute paid to the veterinary staff of the Animal Health Division, and echo the Committee's remark that " care should be taken to ensure that the conditions of service are such as will continue to attract men of a calibre adequate to the discharge of these exacting duties." The conditions of Government service both at home and in the Colonies have for long been a matter of active concern to the B.V.A. It is heartening to see the Association's anxiety for our colleagues in the service of the State shared in so influential a quarter.

Pirbright, too, received its just appreciation. Veterinary surgeons who are concerned with research must often have shared our indignation at the ill-informed reproaches aimed at what the Committee calls " the most important centre in the world for foot-and-mouth disease research." The fact that monovalent and bivalent vaccines have been elaborated which in adult cattle can give 90 per cent. protection is sufficient vindication of the veterinary research staff at Pirbright—if they needed any. It is noteworthy, too, that the Committee, having accepted the view that in the event of an epidemic too widespread for slaughter to be practicable vaccination could be usefully employed, recommend an extension of the manufacturing and storage capacity at Pirbright rather than the production of " stock-piles " of vaccine in other laboratories.

The Report observes, with some justice, that " the history of scientific discovery contains plenty of examples of theories at first rejected by orthodox opinion which eventually established themselves " ; and in this connection evidence was heard from several persons who claimed to have discovered " cures " and preventives which would make slaughter and ordinary vaccination alike unnecessary. It was right and proper, in view of the world-wide concern with the foot-and-mouth problem, that all views should be heard. The conclusion arrived at, however, was that none of these esoteric remedies had anything to commend it above those already in use.

Those who noted the Report's Conclusions and Recommendations will not have failed to observe the Committee's apparently sympathetic interest in the possibility of a Veterinary Research Council analogous in its aims and functions to the Medical Research Council. This is not the place or the time to digress upon a subject with which some of the foremost minds in the profession have for long concerned themselves. Suffice it to say that, if the Committee's hint—should we have interpreted them correctly—gives impetus to this far-reaching and indeed epoch-making possibility they may prove to have rendered no small service to veterinary science in this country, and to have carried a stage further forward that advancement in professional and intellectual prestige which must be the desire of all thoughtful veterinarians.

The latter portion of the Committee's Recommendations includes a wide variety of practical steps that should, in their view, be taken, or made more effective, in dealing with outbreaks of the disease when they actually occur. These include closer supervision over waste foods used for " swill "—a potent source of infection—the prohibition of milk feeding to calves in transit, which was the cause of a disastrous spread of the disease in 1951–52, quicker and more effective publicity to give warning of outbreaks (the *Record* has suffered from lack of prompt information many times in the past just as have practising veterinary surgeons), the control of military manoeuvres and so on. Of particular interest was the suggestion that the fact that members of the profession, especially younger members, have so little opportunity to study foot-and-mouth disease at first hand may be responsible for the occasional failure to detect the disease particularly in atypical cases or very superficial ones. Various methods of making them more familiar with the symptoms were suggested. So far as we are concerned, the *Record* will publish, as soon as it can be prepared, an exhaustive clinical article, illustrated with black and white or coloured plates, in the hope of assisting readers who are in practice to detect the first signs of trouble.

* * * * *

We hope to publish a detailed general article on the Report of the Departmental Committee next week.

FORTHCOMING CONGRESS
THE FEEDING OF FARM LIVESTOCK FOR HEALTH AND PRODUCTION

The B.V.A. is making plans for an Autumn Conference to discuss the feeding of farm livestock for health and production.

It will be held on two consecutive days—Tuesday and Wednesday, November 16th and 17th, 1954—in the South Hall, Victoria Halls, Bloomsbury Square, London, W.C.1.

The Conference will be open to members of the Association and to members of ancillary associations and trade associations. Admission will be by ticket. Arrangements for issue of the tickets will be announced. A full programme will be published in due course.

PULMONARY OEDEMA IN SHEEP CAUSED BY IMMATURE LUNGWORMS

BY

J. F. MICHEL

In the course of our experimental work on *Dictyocaulus filaria* infections in sheep, we have encountered pulmonary oedema on numerous occasions.

In the accompanying table are set forth particulars of ten animals that developed a variable degree of pulmonary oedema and died between 21 and 35 days after infection with lungworm larvae. At *post mortem* examination there was seen to be a variable degree of oedema. At one extreme the lungs were so water-logged that fluid poured out from a cut surface; at the other there was relatively little free oedematous fluid although the lungs were very turgid. These ten animals represent about 10 per cent. of those infected. The number of worms recovered from these cases varied, but their size was fairly constant being approximately 1 cm. long.

In all 10 cases, of which details are given in the table, the animals had failed to show symptoms when seen a few hours before they were found dead. Symptoms of disease have been seen on only one occasion in four Oxford Down lambs which did not die. These developed dyspnoea with rapid abdominal breathing 24 days after being infected with *D. filaria* larvae. The symptoms abated gradually after a week.

In our study of the fog fever syndrome in parasitic bronchitis of cattle, an account of which has already been published, it was concluded that the occurrence of the syndrome is probably associated with the process by which the worms are eliminated. That something similar occurs in sheep is eminently possible for we have found that, especially in older animals, the worms may be eliminated somewhat less than 25 days after first infection.

No.	Date infected	Number of larvae administered	Date of death	Days after infection	Worms recovered post-mortem: Above 1 cm. long	Worms recovered post-mortem: 1 cm. and less
2585	3.8.50	100,000	26.8.50	23	—	34,000
3123	31.5.51	11,400	23.6.51	23	4	1,000
3263	2.4.52	20,000	25.4.52	23	—	6,380
3083	9.8.51	50,000	2.9.50	24	20	1,558
3729	27.7.53	4,000	17.8.53	21	—	2,150
3373	7.8.52	4,000	1.9.52	25	—	1,500
3363	20.10.52	4,000	16.11.52	27	—	372
3752	27.7.53	4,000	24.8.53	28	7	477
3460	20.10.52	4,000	24.11.52	35	—	690
3362	20.10.52	4,000	24.11.52	35	—	19

It will be seen from the table that of the six animals that had received a dose of 4,000 larvae those that died 27 days or more after infection were harbouring considerably fewer worms than were the other two which died 21 and 25 days after infection. We may perhaps postulate that in the animals that died 27 days or more after infection the elimination of the worms was almost complete while in the other two it was only beginning.

It was concluded from these observations that pulmonary oedema should be of relatively frequent occurrence in sheep and it was not long after these observations had been made that news of a naturally occurring outbreak came to hand. Dr. K. C. Sellers, of Leeds, to whom the writer is indebted, came upon an outbreak in which six of 200 lambs had died suddenly with oedematous lungs. The surviving animals at this time showed no symptoms and there was no coughing. Dr. Sellers rejected the tentative diagnosis of pulpy kidney that had been made, and sent to this laboratory the lungs of a poor though apparently unaffected animal that was slaughtered for the purpose. From the lungs of this animal we recovered 55 lungworms of the size found in our experimental oedema cases as described above. Though the number of worms recovered from this lamb was small this animal had not itself been clinically affected and it was considered that the six deaths had probably been due to recent lungworm infection. The circumstances therefore seemed to warrant the suggestion that an outbreak of husk would ensue, and when the farm was visited a fortnight later the predicted symptoms of husk had indeed appeared. All 200 lambs were coughing violently and another lamb killed at this time was found to be harbouring adult lungworms. The grazing history of the lambs suggested that they had picked up a heavy infestation three weeks before the first deaths occurred.

Although this is the only outbreak that has so far come under our observation it does not appear unreasonable to suppose that sudden deaths with pulmonary oedema in lambs may well be a relatively common occurrence and perhaps a frequent prelude to outbreaks of parasitic bronchitis. This note is therefore published in the hope that drawing attention to this condition may lead to an extension of our knowledge of its incidence and of the attending circumstances.

CLINICAL COMMUNICATION

THE OCCURRENCE OF DISEASE IN WILD BIRDS ASSOCIATED WITH *BACT. PARACOLI*

BY

A. McDIARMID

Agricultural Research Council, Field Station, Compton, Berks

The rôle of the "paracolon" group of organisms has not been considered to be of much significance in the causation of disease, but reports have recently begun to appear in the literature incriminating this type of organism as a definite pathogen for man. Kernohan (1952) described a case of septicaemia in a young girl and, in addition, reviewed the recent information pertaining to this subject. He stressed the fact that, within the last few years, other authors had also considered this group of organisms as the causal agents of a variety of gastro-intestinal infections, *e.g.*, Block, Milzer & Kerdeman (1949), and Dornall (1948). He concluded his paper by stating that "the pathogenic rôle of *Bact. paracoli* may not be so limited as previous reports suggest." Subsequently Cronk (1952) stressed its apparent pathogenic effect on man and the ultimate successful treatment of this particular case with chloramphenicol. In animals, Hinshaw & McNeil (1946a) recorded severe mortality in turkeys due to *Bact. paracoli* and suggested the possibility of rattlesnakes acting as reservoir hosts (1946b). The same authors also isolated organisms of the paracolon group from lizards in California (1947).

My interest in studying diseases of free-living wild animals and birds originally arose through attempts to discover possible correlations which might exist between infections in wild life and similar conditions in domestic animals ; so far, two cases of " paracolon " infection have been encountered in the wild bird population and in view of the present interest in these infections a report on these cases appeared warranted.

Clinical History of Cases

Two birds, both in poor condition, were obtained from neighbouring farms in Berkshire. The first was an immature wood-pigeon which had obviously only recently left the nest and this bird was shot as being from previous experience a specimen worth examining. The second was an immature partridge hardly able to fly ; it was left behind by the remainder of the covey, only five in all, a fact that was noted at the time as possibly being of some significance as the majority of coveys in that neighbourhood consisted of at least double this number during that particular season.

Post-mortem Findings

The macroscopic pathological picture in both birds was strikingly similar to bacillary white diarrhoea (B.W.D.) in domestic poultry chicks, the lesions consisting of numerous small yellow-white necrotic foci distributed throughout the myocardium, lungs and liver, the last-named organ being enlarged. Enteritis was present in both specimens.

Histology

Sections prepared from the tissues and stained with haematoxylin and eosin, and by Gram's method showed a disease with a pathological picture closely similar to B.W.D.

Bacteriological Examination

Smears from the lesions of both birds showed numerous gram-negative cocco-bacilli. Pure cultures were readily obtained on MacConkey medium inoculated with material from the infected tissues. The small, grey-white colonies which developed after 48 hours' incubation at 37° C. gave the impression that we were dealing with a clear-cut Salmonella infection. The two strains were accordingly subcultured and dried *in vacuo* pending further investigation. Eventually these dried cultures were reconstituted and their reactions examined in carbohydrate media, containing Andrade's indicator ; it was shown that the two strains differed somewhat in their fermentation reactions and also in the indole and urea tests ; the strains were finally examined by Dr. Joan Taylor at Colindale who suggested that both types of organism belonged to the " paracolon " group. Table I shows some of the bacteriological characteristics of both strains.

Discussion

Three main points may be considered in connection with these two cases. Firstly, a disease caused by " paracolon " organisms can apparently occur in free-living wild birds—namely, the wood-pigeon and partridge. It is feasible that this disease may even act as a limiting factor in the populations concerned and this is supported by the fact that there had probably been deaths in the partridge covey before this bird was obtained. It is well known that a condition resembling B.W.D. has been noticed by gamekeepers in wild partridges and it has been stated by Vesey-Fitzgerald (1946) that B.W.D. should be considered as one of the partridge diseases causing serious loss. Over a number of years we have attempted to secure a case of B.W.D. in this or any other species of wild bird but so far we have been unsuccessful and, although we admit that B.W.D. could conceivably occur in a brood of hand-reared partridges the infection being contracted from the carrier foster-hen, we consider that in view of the findings recorded in this paper it seems worth while to emphasise that a correct diagnosis of B.W.D. cannot be made without laboratory examination to identify the causal organism.

Secondly, in accredited poultry flocks, a small proportion of non-specific reactors to the plate or rapid agglutination test for B.W.D. occur from time to time in adult stock. These cases apparently do not develop clinical symptoms and the usual finding is that such birds are negative when retested 30 days later. It may be that at least a proportion of these peculiar indefinite reactions result from transient infection of the hens with " paracolon " organisms acquired from wild birds. As enteritis was present in our two cases this clearly indicates the strong possibility of a frequent shedding of organisms with consequent contamination of the immediate surroundings. On enquiry it was found that one of the farms from which these specimens were obtained had in fact had reactors of this kind in poultry. Although precise scientific proof is lacking some evidence is available to show that adult poultry inoculated intravenously with suspensions of these organisms may become sensitised albeit for a very short time to the B.W.D. antigen : four adult poultry, negative to the B.W.D. test, were inoculated intravenously with suspensions of paracolon organisms. They reacted indefinitely to this

Table I
Bacteriological Characteristics of *Bact. paracoli* Isolated from Wild Birds

Origin of Strain	Morphology	MacConkey Medium	Ox-Blood Agar	Glucose	Maltose	Mannile	Salicin	Lactose	Sucrose	Sorbitol	Dulcile	Indole	Urea	Serological Reaction
Wood-pigeon Liver	Small gram-negative cocco-bacilli	Small grey-white colonies after 48 hrs. incubation at 37° C.	Non-haemolytic grey-white colonies after 48 hours at 37° C.	A 1 day	A 1 day	A 1 day	A 2 days	—	—	A 2 days	—	—	+	Not agglutinated by any Salmonella sera
Partridge Lung	as above	as above	as above	AG 1 day	AG 1 day	AG 1 day	AG 4 days	—	—	—	—	+	—	Not agglutinated by any Salmonella sera

A signifies Acid
AG „ Acid and gas
— „ No reaction

test about the 15th day after inoculation but were completely negative by the 20th day.

Thirdly, we have the question of the obvious relationship to public health. We believe that the meat inspector, so far as game is concerned, is interested mainly in putrefactive conditions, so that provided the game is not hung too long before being offered for sale it is not detained. Moreover, a considerable quantity of game consumed by the public never passes through the meat inspector's hands.

Summary

(1) " Paracolon " infection has been detected in two species of wild birds and may be of greater significance as a cause of mortality than has been previously thought.

(2) A proportion of the temporary indefinite reactions to the B.W.D. agglutination test met with in accredited flocks of domestic poultry may be associated with this organism.

(3) " Paracolon " infection has been reported in the human subject and in this paper one possible source of this infection is demonstrated.

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ABSTRACT

Zoonoses in Kenya*

In this paper, which was presented to the Kenya branch of the British Medical Association in November, 1953, the author stresses the importance of the two professions jointly concentrating in fields of mutual interest and none more so than in " those diseases of animals transmissible to man " to which the term " zoonoses " is nowadays applied.

In many countries in recent years there have been efforts at bridging the gap—made in the past by unilateral efforts—and to integrate these efforts with the organisation of veterinary public health units within the framework of provincial district and municipal departments.

Tapeworm diseases in the colony cover a wide range and the author selects one as representative, viz., *Taenia saginata* or *inermis*. The actual incidence of taenia infections is unknown but it appears that a drastic cleaning-up campaign is both urgent and essential.

Living conditions and general hygiene especially among town dwellers have improved but the rural population still keep to their old unsanitary habits.

Apart from the economic loss the eradication of *Taenia saginata* is perhaps even more desirable from the public health aspect. General debility, epilepsy and psychoses are some of the troubles caused by its presence in man.

Cysticercosis and other cystic affections also present a problem. The tendency of the larvae to head for a particular muscle is well known and most text-books on meat inspection regard the masseter muscles and the heart as predilection sites. In Kenya, however, the author and his co-workers give priority to the shoulder muscles. For example, out of 358 infected cattle, 102 (26·1 per cent.) showed cysts in the shoulder muscles only, 51 (14·2 per cent.) in the masseter muscles, 26 (7·2 per cent.) in the heart and in only a single case in the diaphragm and flank. The remaining 177 cattle had cysts in two or more places but in the majority of cases in the shoulder muscles as well.

The method of inspection is dwelt upon by the author who shows the need for a more thorough examination of the muscles than obtains in lands where the incidence is low and the general hygienic standards higher.

Any incision into the muscles of a carcase is to a certain extent mutilation and the meat inspector in Britain abstained from this practice for a considerable length of time. *Cysticercus bovis* was observed to be on the increase and inspection methods had to be drastically revised. At present deep incisions into the muscles of mastication form a part of the routine inspection in adult cattle. This procedure, combined with inspection of the heart and exposed carcase muscles may be satisfactory in lands where the incidence is comparatively low and general hygiene high. More precautions are however essential in a country where the percentage is 21 or over and hygienic standards low.

According to most text-books and meat legislation the inspection for cysticercus can be omitted in calves under the age of six weeks but in Kenya, where an average of 56·5 per cent. of young calves are found to be infected, this idea had to be revised. The author considers that hand rearing and unhygienic farms play a large part in the infection of the young.

The sex of the animal is regarded as having some bearing upon the incidence of infection, the male according to some authors always shows a higher percentage of infestation than the female.

In the course of meat inspection the author found that the incidence of cysticercosis in native cattle to be much lower in older animals and considers that this is due to an acquired resistance after several infections.

If one compares the average of 56·5 per cent. found in calves with that of 21 per cent. in adult cattle, a substantial number has cleared itself of cysticercosis. The question is raised as to whether this is an acquired resistance or whether it is due to a degeneration and absorption of the first infection and the transfer to a clean pasture. The author favours the latter view for most of the cattle in which the percentage of infection was small came from ranching estates, leaving the farm at the age of two and remained on the open ranch for from three to four years. On the other hand cattle kept in the vicinity of the farm showed a high percentage of infection. If immunisation had taken place it is the latter group which should show a much smaller percentage of infection being all the time in an infested environment.

The economic aspect of cysticercosis in regard to Kenya is discussed and a loss of over £30,000 annually is mentioned. In actual fact the figure is probably much higher.

The public health aspect is even more serious for as well as the trouble caused by the presence of *Taenia saginata* in man, the carrier of *Taenia solium*, the pig tapeworm, constitutes a still more serious problem as he not only auto-infects his own organism with viable eggs but directly endangers the health of others.

A drastic cleaning-up campaign of *Taenia saginata* and *Cysticercus bovis* is urgent and essential and the two professions have to make a united effort. A campaign has

* **Zoonoses in Kenya.** Ginsberg, A. (1954). *E. Afr. med. J.* **31.** 81–88.

already started in Kenya and all cases of cysticercosis found in cattle at Athi, or pigs at Uplands are reported to the Director of Veterinary Services and the Director of Medical Services. The District Veterinary Officer and District Medical Officer then receives the names of infected farms with a view to improving the sanitation, eliminating the human carrier, and giving advice on methods of prevention and animal hygiene.

The author emphasises the importance of a well established meat inspection service in combating zoonoses.

Meat inspection in Kenya is at present unsatisfactory with only a few municipalities and two meat production plants having a compulsory examination carried out by qualified meat inspectors. He considers that the colony should be divided into meat inspection districts headed by qualified inspectors directly responsible to the central authority. It should be made compulsory that all animals intended for human consumption are inspected.

F.L.

QUESTION IN PARLIAMENT

Myxomatosis

Major Anstruther-Gray (July 26th) asked the Minister of Agriculture if he will give details of the recent spread of myxomatosis among rabbits both North and South of the Border.

Mr. Nugent: Since the reply given to my hon. and gallant Friend on July 15th, isolated outbreaks of the disease have been confirmed for the first time in Hertfordshire, Nottinghamshire, Shropshire, Somerset, Wiltshire, Worcestershire, Denbighshire and Merionethshire. So far as is known, the only outbreak in Scotland is that in Kincardineshire. In most of the areas affected the disease appears to be spreading slowly.

*

NOTES AND NEWS

Diary of Events

Aug. 27th.—Summer Meeting of the Eastern Counties Division, B.V.A., at the Red Lion Hotel, Colchester, 11.45 a.m.

Sept. 19th—25th.—72nd Annual General Meeting and Congress of the British Veterinary Association at Torquay.

Nov. 16th & 17th.—B.V.A. Conference on the Feeding of Farm Livestock for Health and Production in the South Hall, Victoria Halls, Bloomsbury Square, London, W.C.1.

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PERSONAL

Forthcoming Marriage.—Castle—Bailey.—The engagement is announced between Malcolm Edward Castle, B.Sc., Ph.D., younger son of Mr. and Mrs. G. Castle, of Liversedge, Yorkshire, and Elizabeth Joy Bailey, B.V.Sc., only daughter of Mr. and Mrs. G. L. Bailey, of Boxmoor, Herts.

. . . . .

Birth.—Gilbert.—At Barratt Maternity Home, Northampton, to Sheila (née Buck) wife of Gerald Gilbert, M.R.C.V.S.—a son.

* * * * *

R.C.V.S. OBITUARY

Craig, John (Capt., late R.A.V.C.), of Ardlui, East Kilbride, Lanarks. Graduated Edinburgh, December 19th, 1904. Died July 25th, 1954; aged 74 years.

Rix, Clement Eden (Capt. late R.A.V.C.) of 304, Broadway and 55, Danson Road, Bexleyheath, Kent. Graduated London, July 12th, 1899. Died August 2nd, 1954; aged 77 years.

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EUROPEAN COMMITTEE ON FOOT-AND-MOUTH DISEASE

Sir Thomas Dalling, M.A., D.Sc., F.R.C.V.S., F.R.S.E., has been appointed first Secretary of the European Foot-and-Mouth Disease Commission.

* * * * *

ADDRESSES OF DISEASE-INFECTED PREMISES

The list given below indicates, first, the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax:

Derbys.—Haddon House Farm, Haddon Road, Bakewell (July 29).

Leics.—Glebe Farm, Ratcliffe Culey, Atherstone (July 27).

Renfrew.—Bogside Farm, Inverkip, Greenock (July 26).

Fowl Pest:

Essex.—Coneygrey, Nevendon Road, Wickford; Heathfields, Fordham Heath, Lexden, Colchester (July 24); Newlands Farm, Eight Ash Green, Colchester (July 27).

Lancs.—Bridge Farm, Laneshaw Bridge, Colne (July 27).

Leics.—Welby Lane, Ashfordby Valley, Melton Mowbray (July 24); Debdale Farm, Thorpe Arnold, Melton Mowbray (July 25); Sidegate Farm, Barwell (July 26); 102, Colby Drive, Thurmaston; Red Hall Farm, Barwell (July 27); The Cow Pastures, Welby House Farm, Welby, Ashfordby, Melton Mowbray (July 29).

Swine Fever:

Berks.—Princess Manor Farm, Harwell, Didcot (July 27).

Ches.—Lower Carr Green Farm, Warburton (July 26).

Cornwall.—Pencrebar Farm, Collington (July 29).

Devon.—Lobb Farm, Hemerdon, Plympton, Plymouth (July 28); Commonwood House, off Plymbridge Road, Riverford, Roborough, Plymouth (July 29).

Dunbarton.—Edinbarnet Stables, Duntocher, Glasgow (July 26).

Glamorgan.—Cefn Carnau Fach Farm, Caerphilly (July 26).

Glos.—Post Office, Easter Compton, Bristol (July 30).

Gloucs.—Nupend Post Office, Nupend, Stonehouse (July 26).

Hunts.—Hollow Heap Farm, Ramsey, Huntingdon (July 27).

Lancs.—Walton Hall Farm, Walton le Dale, Preston; Proctors Farm, Pilling Lane, Preesall, Fleetwood; Home Lea Farm, White Horse Lane, Myerscough, Garstang, Preston (July 26); Slate Lane Piggeries, Audenshaw, Manchester (July 28); Hanover Street Piggeries, Audenshaw, Manchester (July 30).

Lincs.—Station Road, Surfleet, Spalding (July 26); West End Farm, Winteringham, Scunthorpe (July 27); Sandbraes Farm, Caistor, Lincoln (July 29); Chestnut Farm, Sutton Bridge, Spalding (July 30).

Norfolk.—Fritton Farm, Ludham, Great Yarmouth (July 26); New Road Premises, Upwell; Pond Farm, Roughton, Norwich (July 27).

Notts.—Grange Farm, Barnby, Newark (July 30).

Oxford.—95, High Street, Kidlington (July 28).

Salop.—Newtown Farmhouse, Baschurch, Shrewsbury (July 26); Orchard Cottage, Halfway House, Westbury, Shrewsbury (July 27); Fields Farm, Alkington, Whitchurch (July 28); Park Farm, Yockleton, Shrewsbury (July 28).

Somerset.—Hanging Grove Farm, Winford, Bristol (July 27).

Staffs.—Yew Tree House, Tatenhill Common, Burton-on-Trent (July 26); The Cottage, Salt, Stafford (July 27).

Suffolk.—Street Farm, Stowlangtoft; Bridge Farm, Stowlangtoft, Bury St. Edmunds (July 27).

Sussex.—Manor Farm, Tarring Neville, Newhaven (July 27).

Warwicks.—Gilsend Hall Farm, Gilson, Coleshill, Birmingham (July 27).

Worcs.—Canal Side Cottage, Whitford Bridge, Stoke Pound, Bromsgrove (July 26).

Yorks.—Withgill, Clitheroe (July 26).

CORRESPONDENCE

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

SKIN CONDITIONS IN THE DOG

Sir,—I have read the Clinical Communication by Messrs. A. H. Hogg and H. Holroyd with great interest. I have seen both conditions over a period of years, and to the breeds mentioned I would like to add the Irish setter and the golden labrador.

I have used various treatments but not the vitamin " B " complex* for the first condition described; iodine oil gives very good results. Benzyl benzoate for the tail lesion is useful but the condition tends to re-appear six months or a year later.

Further correspondence would be welcome on this interesting subject.—Yours faithfully, G. L. B. Henderson, 30, Claremont Road, Surbiton. *July 25th*, 1954.

* The reference to " Becosyn " (Roche Products), in the article by Messrs. A. H. Hogg and H. Holroyd, should be corrected to " Becosym " (Roche Products) which is a vitamin " B " complex. Authors and correspondents should note that wherever possible it is the policy of the *Record* to publish the official description of all products, indicating the trade names at the foot of the contribution.—*Editor.*

* * * * *

BIOLOGICAL APPROACH TO MEDICAL GEOGRAPHY

Sir,—In *The Veterinary Record* for July 24th, a paper by J. R. Audy—" A Biological Approach to Medical Geography "—is reviewed by I. W. J. who states " For those interested in preventive medicine the author suggests a new approach to the subject of medical geography, namely the study of the whole assemblage of parasites in their relationship with the host population. The parasite patterns which emerge from such a synecological approach are considered to be decided largely by environment—that is, by the geographical background in the case of animals."

May I suggest that this concept can scarcely be considered as new? As long ago as November, 1951, I published a paper in the *British Veterinary Journal* entitled " The Ecology of Infectious Disease," which stressed the vital importance of the ecological approach in the study of the host-parasite relationships of infectious disease in particular, though of course it is equally true of parasitic infestations. The environment of an organism is provided by three factors, climate, soil and other organisms, in that order of importance. Climate and soil are provided for a parasite by the temperature of the host and the physico-chemical composition of the host tissues.

Throughout its life an organism is dominated by these factors, and the pathogenic parasite is no exception. I claim no particular credit for my paper, for although it contains observations of my own, it is buttressed by references to authorities who have worked on similar lines. The supreme importance of the ecological approach has already been grasped in biological fields, and mycological research, for example, now focuses on all data relating to the influence of climate and soil on the life cycle of phyto-parasites. Markham drew attention in 1947 to the influence of topography and climate upon infant mortality and death-rate. Havilland wrote his classic treatise upon medical geography as long ago as 1875, and amongst other observations comments upon the association of rheumatism (which may be bacterial in origin) with valleys and water courses. More recently, Lovell records that an extensive study of the nasopharyngeal bacterial flora of different groups of persons in London and South-east England showed a seasonal trend associated with increasing sunshine and decreasing relative humidity.

All credit is due to Audy for his careful study and the conclusions he has drawn. He must be congratulated for his contribution to a field which, as indicated, has not been quite overlooked, but which merits more serious attention than is at present spared by professional circles.

I suggest, sir, that many pathological problems might be solved if we ceased typing the laboratory reactions of parasites under controlled conditions, and got out into the fresh air to see whether the environment provided by the host under field conditions was suitable or not.—Yours faithfully, Ernest A. Gray, Chief Bacteriologist, Bayer Biological Institute, Exning, Nr. Newmarket, Suffolk. *July 29th*, 1954.

References

Audy, J. R. (1954). *Brit. Med. J.* 960.

Gray, E. A. *Brit. Vet. J.* **107.** 441.

Markham, S. F. (1947). " Climate and the Energy of Nations." London.

Havilland, A. (1875). "Geographical Distribution of Heart Disease, Dropsy, Cancer in Females and Phthisis in Females." London.

Lovell, R. (1950). *Proc. Roy. Soc. Med.* **43.** 1.

DISEASES OF ANIMALS ACT, 1950—GREAT BRITAIN

Summary of Returns of Confirmed Outbreaks of Scheduled (Notifiable) Diseases

Period	Anthrax	Atrophic Rhinitis	Foot-and-mouth	Fowl Pest	Sheep Scab	Swine Fever
1st to 15th July, 1954 ...	10	—	—	9	—	65
Corresponding period in—						
1953 ...	11	—	—	111	—	93
1952 ...	36	—	29	5	—	22
1951 ...	7	—	3	22	—	32
1st January to 15th July, 1954	160	3	12	550	—	889
Corresponding period in—						
1953 ...	445	—	26	619	—	1,826
1952 ...	310	—	437	126	1	579
1951 ...	267	—	21	516	4	510

Tuberculosis Order of 1938

The number of bovine animals slaughtered under the tuberculosis Order of 1938 during the period April 1st to June 30th, 1954, was 508.

Advertiser's Announcement

Genatosan Limited

" Braxy and Blackleg Vaccine A.P.S.," which has superseded " Braxy and Blackleg Vaccine," is now available for sale. It is packed in bottles of 50 c.c., 100 c.c., and 250 c.c., and it should be noted that the dosage of " Braxy and Blackleg Vaccine A.P.A." for sheep is 2 c.c. as compared with a dosage of 5 c.c. for the original " Braxy and Blackleg Vaccine " and the dosage for cattle is 5 c.c. instead of 10 c.c.

" C. Pyogenes Toxoid " is now being produced in 50 c.c. and 100 c.c. bottles. At the moment there are warehouse stocks of 50 c.c. bottles, and 100 c.c. bottles will be available shortly.